Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgment before publication.

Fallacies and PPI's

TO THE EDITOR: Dr. Goyan's comments on patient package inserts (PPI)[1] in the May 1981 issue were interesting, and I agree with most of them. However, certain points need clarifying.

The third fallacy (patients don't need PPI information), of the 14 he discusses, has little to do with whether patients' compliance will be enhanced by PPI's. Many doctors feel that PPI's will *decrease* patient compliance by frightening them away from taking medicine. Who would take aspirin after reading a complete list of possible side effects, including bleeding, ulcer and life-threatening anaphylaxis? Patients want information on how to take drugs (with or without food, with or without other medicines, AM or PM) and common side effects. But what purpose is served by listing dozens of reactions that are rare, are only occasional or occur in less then 1 percent of cases? And how could a physician prescribe a medication for a purpose not listed on the PPI? All he would hear would be, "Why are you giving me an arthritis drug for menstrual cramps?" or "This drug treats *Trichomonas,* not *Giardia,* so it can't do me any good."

Dr. Goyan's fallacy is that patients are as sophisticated as doctors in their ability to sift through drug information in PPI's. Physicians must frequently edit information for their patients to put it in a form they can understand and accept.

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REFERENCE

1. Goyan J: Fourteen fallacies about patient package inserts (Informed Opinion). West J Med 134:463-468, May 1981

Drugs Associated With Retroperitoneal Fibrosis

TO THE EDITOR: I read with interest "Retroperitoneal Fibrosis as a Cause of Fever of Undetermined Origin" in the April issue.[1] In 1948 idiopathic retroperitoneal fibrosis was first described as a clinical entity. Most often the patient is a middle-aged man with primary symptoms of backache, anorexia and weight loss in whom oliguria and azotemia subsequently develop.[2] This symptom complex is seen with fibrous tissue growth in the retroperitoneal space which encroaches upon nearby organs and blood vessels. The fibrous tissue appears as a glistening, white, woody hard plaque.

Dr. Byrd's case description is atypical in many respects. Abdominal or back pain, absent in this patient, occurs in most published cases. The localized fibrous tissue mass without ureteral involvement is not characteristic. Usually one or both ureters are encased by contracting fibrous tissue which is usually confined to the limits of Gerota's fascia.[3] Most reports of this disorder are found in the urologic literature because patients always present with ureteral obstruction and frequent azotemia, and are quickly referred to a urologist. Retrograde placement of ureteral catheters is easily accomplished in spite of the fibrosis surrounding the ureters. Surgical lysis and peritonealization of the ureters reverses the azotemia and frequently relieves the pain. Although a hypochromic anemia is characteristic in idiopathic retroperitoneal fibrosis, it is usually not so severe as to require transfusions.

Many pharmacologic agents have been associated with the development of retroperitoneal fibrosis: methylsergide, hydralazine,[4] ergotamine tartrate,[5] methyldopa,[6,7] aspirin-phenacetin-codeine analgesics,[8,9] oxyprenolol,[10] atenolol,[11,12] lysergic acid diethylamide (LSD),[5] dextroamphetamine, ephedrine and strychnine.[3] Since this list of drugs appears to involve vasoactive substances, it has led to a hypothetical pathogenic mechanism. The prolonged and repeated arteriolar vasoconstrictions and vasodilations may lead to perivascular edema, transudation of plasma content and the initiation of the sustaining fibroblastic response.[3] We must realize the possibility that many drugs commonly prescribed may conjugate with antigens, incite a hypersensitivity response and lead to retroperitoneal fibrosis. Since retroperitoneal